Paper Alert

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A selection of interesting papers that were published in the month before our press date in major journals most likely to report significant results in structural biology, protein and RNA folding.

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Structure 2000, **8**:R189–R193

☐ **Structural basis for the interaction between FxFG nucleoporin repeats and importin-β in nuclear trafficking.** Richard Bayliss, Trevor Littlewood and Murray Stewart (2000). *Cell* **102**, 99–108.

The authors describe the crystal structure of a complex between importin-β residues 1–442 (Ib442) and five FxFG nucleoporin repeats from Nsp1p. Nucleoporin FxFG cores bind on the convex face of Ib442 to a primary site between the A helices of HEAT repeats 5 and 6, and to a secondary site between HEAT repeats 6 and 7. Mutations at Ile178 in importin-β, a primary FxFG-binding site, reduce both binding and nuclear protein import. These FxFG-binding sites on importin-β do not overlap with the RanGTP-binding site.
7 July 2000, *Cell*

☐ **Crystal structure of the worm NitFhit Rosetta Stone protein reveals a Nit tetramer binding two Fhit dimers.** HC Pace, SC Hodawadekar, A Draganescu, J Huang, P Bieganowski, Y Pekarsky, CM Croce and C Brenner (2000). *Curr. Biol.* **10**, 907–917.

The nucleotide-binding protein Fhit suppresses tumor formation by inducing apoptosis. In invertebrates, Fhit is encoded as a fusion protein with Nit, a member of the nitrilase superfamily. The crystal structure of Nit–Fhit shows that Nit monomers possess a new α–β–β–α sandwich fold with a presumptive Cys–Glu–Lys catalytic triad. Nit assembles into a tetrameric, 52-stranded β box that binds Fhit dimers at opposite poles and displays Nit active sites around the middle of the complex. The most C-terminal β strand of each Nit monomer exits the core of the Nit tetramer and interacts with Fhit.
13 July 2000, *Current Biology*

☐ **The bacterial cell-division protein ZipA and its interaction with an FtsZ fragment revealed by X-ray crystallography.** Lidia Mosyak, Yan Zhang, Elizabeth Glasfeld, Steve Haney, Mark Stahl, Jasbir Seehra and William S Somers (2000). *EMBO J.* **19**, 3179–3191.

In *Escherichia coli*, FtsZ, a homologue of eukaryotic tubulins, and ZipA, a membrane-anchored protein that binds to FtsZ, are two essential components of the septal ring structure that mediates cell division. The X-ray crystal structures of the C-terminal FtsZ-binding domain of ZipA and a complex between this domain and a C-terminal fragment of FtsZ are present. The ZipA domain is a six-stranded β sheet packed against three α helices and contains the split β–α–β motif found in many RNA-binding proteins. The uncovered side of the sheet incorporates a shallow hydrophobic cavity exposed to solvent. In the complex, the 17-residue FtsZ fragment occupies this entire cavity of ZipA and binds as an extended β strand followed by an α helix.
3 July 2000, *EMBO Journal*

☐ **Structure of the active core of human stem cell factor and analysis of binding to its receptor Kit.** Xuliang Jiang, Ogan Gurel, Elizabeth A Mendiaz, George W Stearns, Christi L Clogston, Hsieng S Lu, Timothy D Osslund, Rashid S Syed, Keith E Langley and Wayne A Hendrickson (2000). *EMBO J.* **19**, 3192–3203.

Stem cell factor (SCF) is an early-acting hematopoietic cytokine that elicits multiple biological effects. SCF is dimeric and transduces signals by ligand-mediated dimerization of its receptor, Kit. The crystal structure of SCF has characteristic helical cytokine topology, but the structure is unique apart from core portions. The SCF dimer has a symmetric 'head-to-head' association. A model for the SCF–Kit complex is proposed by analogy with related receptors. Similar results have been reported by Zhang *et al.*, (*Proc. Natl Acad. Sci. USA* **97**, 7732–7737).
3 July 2000, *EMBO Journal*

☐ **A new crystal form for the dodecamer C-G-C-G-A-A-T-T-C-G-C-G: symmetry effects on sequence-dependent DNA structure.** Eric Johansson, Gary Parkinson and Stephen Neidle (2000). *J. Mol. Biol.* **300**, 551–561.

This dodecanucleotide has been crystallised in the space group $P3_212$, representing a new crystal form for this sequence, and refined at 1.8 Å resolution. The present structure contrasts with previous ones for this sequence because it is situated on a crystallographic twofold axis, and the crystal symmetry reflects the palindromic nature of this sequence. Some features are consistent with previous observations, notably that the minor groove is hydrated with a continuous spine of solvent. Various base and base-pair morphological parameters have been examined. Their values do not show significant correlations with earlier reports, suggesting that crystal-packing effects are more dominant than has been hitherto realised.
6 July 2000, *Journal of Molecular Biology*

☐ **Solution structure of the cysteine-rich domain of the *Escherichia coli* chaperone protein DnaJ.** Maria Martinez-Yamout, Glen B Legge, Ouwen Zhang, Peter E Wright and H Jane Dyson (2000). *J. Mol. Biol.* **300**, 805–818.

The solution structure of the cysteine-rich (CR) domain of *Escherichia coli* DnaJ has been solved by NMR methods. The structure of a 79-residue CR domain construct shows a novel fold with an overall V-shaped extended β-hairpin topology. The CR domain is characterized by four CxxCxGxG sequence motifs that bind two zinc ions. Residues in these two zinc modules show strong similarities in the grouping of resonances in the ^{15}N–^{1}H HSQC spectrum and display pseudo-symmetry of the motifs in the calculated structures. The conformation of the cysteine residues coordinated to the zinc ion resembles that of the rubredoxin knuckle, but there are significant differences in hydrogen-bonding patterns in the two motifs. Zinc ^{15}N–^{1}H HSQC titrations indicate that the fold of the isolated DnaJ CR domain is zinc-dependent and that one zinc module folds before the other. The CxxCxGxG sequence motif is highly conserved in CR domains from a wide variety of species. The three-dimensional structure of the *E. coli* CR domain indicates that this sequence conservation is likely to result in a conserved structural motif.

21 July 2000, *Journal of Molecular Biology*

☐ **Activation of human liver glycogen phosphorylase by alteration of the secondary structure and packing of the catalytic core.** Virginia L Rath, Mark Ammirati, Peter K LeMotte, Kimberly F Fennell, Mahmoud N Mansour, Dennis E Danley, Thomas R Hynes, Gayle K Schulte, David J Wasilko and Jayvardhan Pandit (2000). *Mol. Cell* **6**, 139–148.

Glycogen phosphorylases catalyze the breakdown of glycogen to glucose-1-phosphate, which enters glycolysis to fulfill the energetic requirements of the organism. The authors have determined the crystal structures of the active and inactive forms of human liver glycogen phosphorylase *a*. During activation, 40 residues of the catalytic site undergo order/disorder transitions, changes in secondary structure, or packing to reorganize the catalytic site for substrate binding and catalysis. Knowing the inactive and active conformations of the liver enzyme and how each differs from its counterpart in muscle phosphorylase provides the basis for designing inhibitors that bind preferentially to the inactive conformation of the liver isozyme.

July 2000, *Molecular Cell*

☐ **Crystal structure of the DNA-binding domain of the replication initiation protein E1 from Papillomavirus.** Eric J Enemark, Grace Chen, Daniel E Vaughn, Arne Stenlund and Leemor Joshua-Tor (2000). *Mol. Cell* **6**, 149–158.

Papillomaviral infection causes both benign and malignant lesions, and many cervical cancers result from such infections. Replication of this virus requires the replication initiation proteins E1 and E2, which bind cooperatively at the origin of replication (*ori*) as an (E1)2–(E2)2–DNA complex. The authors present the crystal structure of the E1 DNA-binding domain refined to 1.9 Å resolution. Residues critical for DNA binding are located on an extended loop and an α helix. The E1 dimerization surface was identified by selective mutations at an E1–E1 interface observed in the crystal.

July 2000, *Molecular Cell*

☐ **Crystal structure and mutagenic analysis of the inhibitor-of-apoptosis protein survivin.** Steven W Muchmore, Jun Chen, Clarissa Jakob, Dorothy Zakula, Edmund D Matayoshi, Wei Wu, Haichao Zhang, Fengzhi Li, Shi-Chung Ng and Dario C Altieri (2000). *Mol. Cell* **6**, 173–182.

The coupling of apoptosis (programmed cell death) to the cell division cycle is essential for homeostasis and genomic integrity. The authors report the crystal structure of survivin, an inhibitor of apoptosis, which has been implicated in both control of cell death and regulation of cell division. In addition to a conserved N-terminal Zn finger baculovirus IAP repeat, survivin forms a dimer through a symmetric interaction with an intermolecularly bound Zn atom located along the molecular dyad axis. The interaction of the dimer-related C-terminal α helices forms an extended surface of ~70 Å in length. Mutagenesis analysis revealed that survivin dimerization and an extended negatively charged surface surrounding Asp71 are required to counteract apoptosis and preserve ploidy. Similar results are reported by Chantalat *et al.*, in the same issue (*Mol. Cell* **6**, 183–189) and by Verdecia *et al.*, (*Nat. Struct. Biol.* **7**, 602–608).

July 2000, *Molecular Cell*

☐ **Structure of the dimerized hormone-binding domain of a guanylyl-cyclase-coupled receptor.** Focco van den Akker, Xiaolun Zhang, Masaru Miyagi, Xuewen Huo, Kunio S Misono and Vivien C Yee (2000). *Nature* **406**, 101–104.

The atrial natriuretic peptide (ANP) hormone is secreted by the heart in response to an increase in blood pressure. In the crystal structure of the glycosylated dimerized hormone-binding domain of the ANP receptor, the monomer comprises two interconnected subdomains, each encompassing a central β sheet flanked by α helices, and exhibits the type I periplasmic binding protein fold. Dimerization is mediated by the juxtaposition of four parallel helices, which brings the two protruding C termini into close relative proximity. The ANP-binding site maps to the side of the dimer crevice and extends to near the dimer interface. Mechanisms for hormone activation and the allostery of the ANP receptor are proposed.

6 July 2000, *Nature*

☐ **Structure of the Fc fragment of human IgE bound to its high-affinity receptor FcεRIα.** Scott C Garman, Beth A Wurzburg, Svetlana S Tarchevskaya, Jean-Pierre Kinet and Theodore S Jardetzky (2000). *Nature* **406**, 259–266.

The initiation of immunoglobulin E (IgE) mediated allergic responses requires the binding of IgE antibody to its high-affinity receptor, FcεRI. The interaction of the crystallizable fragment (Fc) of IgE (IgE-Fc) with FcεRI involves the extracellular domains of the FcεRIα chain. The crystal structure of

the human IgE-Fc–FcεRIα complex reveals that one receptor binds one dimeric IgE-Fc molecule asymmetrically through interactions at two sites, each involving one Cε3 domain of the IgE-Fc. Similar results are described by Sondermann *et al*. for a human IgG-Fc fragment and its cognate receptor (*Nature* **406**, 267–273).
20 July 2000, *Nature*

☐ **Mimicry of ice structure by surface hydroxyls and water of a β helix antifreeze protein.** Yih-Cherng Liou, Ante Tocilj, Peter L Davies and Zongchao Jia (2000). *Nature* **406**, 322–324.
Insect antifreeze proteins (AFPs) are much more effective than fish AFPs at depressing solution freezing points by ice-growth inhibition. The AFP from the beetle *Tenebrio molitor* is a small protein (8.4 kDa) composed of tandem 12-residue repeats (TCTxSxxCxxAx). Its 1.4 Å resolution crystal structure shows that this repetitive sequence adopts an exceptionally regular β helix. On the conserved side of the protein, Thr–Cys–Thr motifs are arrayed to form a flat β sheet, the putative ice-binding surface. The threonine sidechains have exactly the same rotameric conformation and the spacing between OH groups is a near-perfect match to the ice lattice. Similar results are reported by Graether *et al*. (*Nature* **406**, 325–328).
20 July 2000, *Nature*

☐ **Crystal structure of human homogentisate dioxygenase.** Greg P Titus, Heather A Mueller, John Burgner, Santiago Rodríguez de Córdoba, Miguel A Peñalva and David E Timm (2000). *Nat. Struct. Biol.* **7**, 542–546.
Homogentisate dioxygenase (HGO) cleaves the aromatic ring during the metabolic degradation of phenylalanine and tyrosine. HGO deficiency causes alkaptonuria (AKU). The authors report the first crystal structure of a eukaryotic dioxygenase that catalyzes an aromatic ring-cleavage reaction. Crystal structures of apo-HGO and HGO containing an iron ion have been determined. The HGO protomer, which contains a 280-residue N-terminal domain and a 140-residue C-terminal domain, associates as a hexamer arranged as a dimer of trimers. The active-site iron is coordinated near the interface between subunits in the HGO trimer by a glutamate and two histidine sidechains. HGO represents a new structural class of dioxygenases.
July 2000, *Nature Structural Biology*

☐ **Crystal structure of the *Escherichia coli* thioesterase II, a homolog of the human Nef-binding enzyme.** Jia Li, Urszula Derewenda, Zbigniew Dauter, Stuart Smith and Zygmunt S Derewenda (2000). *Nat. Struct. Biol.* **7**, 555–559.
The authors report the crystal structure of the *Escherichia coli* medium chain length acyl-CoA thioesterase II. This enzyme is a close homolog of the human protein that interacts with the product of the HIV-1 Nef gene, sharing 45% amino acid sequence identity with it. The structure of the *E. coli* thioesterase II reveals a new tertiary fold, a 'double hot dog', showing an internal repeat with a basic unit that is structurally similar to the recently described β-hydroxydecanoyl thiol ester dehydrase. The catalytic site, inferred from the crystal structure and verified by site-directed mutagenesis, involves novel chemistry and includes Asp204, Gln278 and Thr228, which synergistically activate a nucleophilic water molecule.
July 2000, *Nature Structural Biology*

☐ **Crystal structure of the bacterial conjugation repressor FinO.** Alexandru F Ghetu, Michael J Gubbins, Laura S Frost and JN Mark Glover (2000). *Nat. Struct. Biol.* **7**, 565–569.
The conjugative transfer of F-like plasmids is repressed by FinO, an RNA-binding protein. FinO interacts with the F-plasmid-encoded *traJ* mRNA and its antisense RNA. The authors present the crystal structure of FinO, lacking its flexible N-terminal extension. FinO adopts a novel, elongated, largely helical conformation. An N-terminal region, previously shown to contact RNA, forms a positively charged α helix (helix 1) that protrudes 45 Å from the central core of FinO. A C-terminal region of FinO that is implicated in RNA interactions also extends out from the central body of the protein, adopting a helical conformation and packing against the base of the N-terminal helix. A highly positively charged patch on the surface of the FinO core may present another RNA-binding surface. The authors propose that the flexible N-terminal region and the N terminus of helix 1 interact with and stabilize paired, complementary RNA loops in a kissing complex.
July 2000, *Nature Structural Biology*

☐ **Critical role of β-hairpin formation in protein G folding.** Erika L McCallister, Eric Alm and David Baker (2000). *Nat. Struct. Biol.* **7**, 669–673.
Comparison of the folding mechanisms of proteins with similar structures but very different sequences can provide fundamental insights into the determinants of protein folding mechanisms. Despite very little sequence similarity, the 60-residue IgG-binding domains of protein G and protein L both consist of a single helix packed against a four-stranded sheet formed by two symmetrically disposed β hairpins. We demonstrate that, as in the case of protein L, one of the two β turns of protein G is formed and the other disrupted in the folding transition state. Unlike protein L, however, in protein G it is the second β turn that is formed in the folding transition-state ensemble. Substitution of an aspartate residue by alanine in protein G that eliminates an i,i+2 sidechain–mainchain hydrogen bond in the second β turn slows the folding rate 20-fold but has virtually no effect on the unfolding rate. Taken together with previous results, these findings suggest that the presence of an intact β-turn in the folding transition state is a consequence of the overall topology of protein L and protein G, but the particular hairpin that is formed is determined by the detailed

interatomic interactions that determine the free energies of formation of the isolated β hairpins.
August 2000, *Nature Structural Biology*

☐ **Conservation of folding pathways in evolutionarily distant globin sequences.** Chiaki Nishimura, Stefan Prytulla, H Jane Dyson and Peter E Wright (2000). *Nat. Struct. Biol.* **7**, 679–686.
To test the hypothesis that the folding pathways of evolutionarily related proteins with similar three-dimensional structures but widely different sequences should be similar, the folding pathway of apoleghemoglobin has been characterized using stopped-flow circular dichroism, heteronuclear NMR pulse-labeling techniques, and mass spectrometry. The pathway of folding was found to differ significantly from that of a protein of the same family, apomyoglobin, although both proteins appear to fold through helical burst phase intermediates. For leghemoglobin, the burst phase intermediate exhibits stable helical structure in the G and H helices, together with a small region in the center of the E helix. The A and B helices are not stabilized until later stages of the folding process. The structure of the burst phase folding intermediate thus differs from that of apomyoglobin, in which stable helical structure is formed in the A, B, G and H helix regions.
August 2000, *Nature Structural Biology*

☐ **Crystal structure of the HIV-1 integrase catalytic core and C-terminal domains: a model for viral DNA binding.** Julian C-H Chen, Jolanta Krucinski, Larry JW Miercke, Janet S Finer-Moore, Ann H Tang, Andrew D Leavitt and Robert M Stroud (2000). *Proc. Natl Acad. Sci. USA* **97**, 8233–8238.
Insolubility of full-length HIV-1 integrase (IN) limited previous structure analyses to individual domains. By introducing five point mutations, the authors engineered a more soluble IN that allowed generation of multidomain HIV-1 IN crystals. The first multidomain HIV-1 IN structure is reported. It incorporates the catalytic core and C-terminal domains (residues 52–288). The structure is a Y-shaped dimer. Within the dimer, the catalytic core domains form the only dimer interface, and the C-terminal domains are located 55 Å apart. A 26 amino acid α helix, α6, links the C-terminal domain to the catalytic core. A kink in one of the two α6 helices occurs near a known proteolytic site, suggesting that it may act as a flexible elbow to reorient the domains during the integration process. A strip of positively charged amino acids contributed by both monomers emerges from each active site of the dimer, suggesting a minimally dimeric platform for binding each viral DNA end. The crystal structure of the isolated catalytic core domain (residues 52–210), independently determined at 1.6 Å resolution, is identical to the core domain within the two-domain 52–288 structure.
18 July 2000, *Proceedings of the National Academy of Science USA*

☐ **Crystal structure of the Holliday junction DNA in complex with a single RuvA tetramer.** Mariko Ariyoshi, Tatsuya Nishino, Hiroshi Iwasaki, Hideo Shinagawa and Kosuke Morikawa (2000). *Proc. Natl Acad. Sci. USA* **97**, 8257–8262.
In the major pathway of homologous DNA recombination in prokaryotic cells, the Holliday junction intermediate is processed through its association with RuvA, RuvB and RuvC proteins. Specific binding of the RuvA tetramer to the Holliday junction is required for the RuvB motor protein to be loaded onto the junction DNA, and the RuvAB complex drives the ATP-dependent branch migration. The authors have solved the crystal structure of the Holliday junction bound to a single *Escherichia coli* RuvA tetramer. In this complex, one side of DNA is accessible for cleavage by RuvC resolvase at the junction center. The refined junction DNA structure revealed an open concave architecture with a fourfold symmetry. Each arm, with B-form DNA, in the Holliday junction is predominantly recognized in the minor groove through hydrogen bonds with two repeated helix-hairpin-helix motifs of each RuvA subunit.
18 July 2000, *Proceedings of the National Academy of Science USA*

☐ **A systematic exploration of the influence of the protein stability on amyloid fibril formation *in vitro*.** Marina Ramírez-Alvarado, Jane S Merkel and Lynne Regan (2000). *Proc. Natl Acad. Sci.* **97**, 8979–8984.
There are a number of diseases in which normally soluble proteins associate into regular, insoluble amyloid fibrils. The development of *in vitro* model systems in which detailed structural, kinetic, and thermodynamic characterization are feasible is of critical importance to our understanding of the amyloid fibril phenomenon. The formation of amyloid fibrils by proteins that are not associated with disease has been recently described, suggesting that this may be a common property of many proteins and not only of the few proteins associated with amyloidoses. The B1 Ig-binding domain of protein G (B1) is an extremely well-characterized model system. Under certain experimental conditions, some variants of B1 form fibrils with high reproducibility. By controlling the stability of the protein either by mutations or by changing experimental conditions the ability of the protein to form fibrils can be modulated. For all of the variants, the key requirement for fibril formation is to choose conditions in which the population of intermediate conformations present during the unfolding transition is maximized.
1 August 2000, *Proceedings of the National Academy of Science USA*

☐ **Three-dimensional structure of the Tn5 synaptic complex transposition intermediate.** Douglas R Davies, Igor Y Goryshin, William S Reznikoff and Ivan Rayment (2000). *Science* **289**, 77–85.
Genomic evolution has been profoundly influenced by DNA transposition, a process whereby defined DNA segments

move freely about the genome. The authors report the three-dimensional structure of prokaryotic Tn5 transposase complexed with Tn5 transposon end DNA. The molecular assembly is dimeric, where each double-stranded DNA molecule is bound by both protein subunits, orienting the transposon ends into the active sites. This structure provides a molecular framework for understanding many aspects of transposition, including the binding of transposon end DNA by one subunit and cleavage by a second, cleavage of two strands of DNA by a single active site via a hairpin intermediate, and strand transfer into target DNA.
7 July 2000, *Science*

☐ **Structure of the cytoplasmic β subunit – T1 assembly of voltage-dependent K^+ channels.** Jacqueline M Gulbis, Ming Zhou, Sabine Mann and Roderick MacKinnon (2000). *Science* **289**, 123–127.
The structure of the cytoplasmic assembly of voltage-dependent K^+ channels is reported at 2.1 Å resolution. The assembly includes the cytoplasmic (T1) domain of the integral membrane α subunit together with the oxidoreductase β subunit in a fourfold symmetric T14β4 complex. An electrophysiological assay showed that this complex is oriented with four T1 domains facing the transmembrane pore and four β subunits facing the cytoplasm. The transmembrane pore communicates with the cytoplasm through lateral, negatively charged openings above the T14β4 complex. The inactivation peptides of voltage-dependent K^+ channels reach their site of action by entering these openings.
7 July 2000, *Science*

☐ **Towards a structural understanding of Friedreich's ataxia: the solution structure of frataxin.** Giovanni Musco, Günter Stier, Bernhard Kolmerer, Salvatore Adinolfi, Stephen Martin, Tom Frenkiel, Toby Gibson and Annalisa Pastore (2000). *Structure* **8**, 695–707.
Lesions in the gene for frataxin, a nuclear-encoded mitochondrial protein, cause the recessively inherited condition Friedreich's ataxia. Recombinant frataxin behaves in solution as a monodisperse species that is composed of a 15-residue-long unstructured N terminus and an evolutionarily conserved C-terminal domain. The structure of the C-terminal domain consists of a stable seven-stranded antiparallel β sheet packing against a pair of parallel helices. Exposed conserved residues cluster on the β-sheet face of the structure, suggesting that this is a functionally important surface. The absence of all the features expected for an iron-binding activity strongly support an indirect involvement of frataxin in iron metabolism.
15 June 2000, *Structure*

☐ **Insights into molybdenum cofactor deficiency provided by the crystal structure of the molybdenum cofactor biosynthesis protein MoaC.** Margot M Wuebbens, Michael TW Liu, KV Rajagopalan and Hermann Schindelin (2000). *Structure* **8**, 709–718.
The molybdenum cofactor (Moco) is an essential component of a large family of enzymes involved in important transformations in carbon, nitrogen and sulfur metabolism. The crystal structure of MoaC from *Escherichia coli* reveals a tightly packed hexamer with 32 symmetry. The monomer belongs to the ferredoxin family. Analysis of structural and biochemical data strongly suggests that the active site is located at the interface of two monomers.
15 July 2000, *Structure*

☐ **Crystal structure of *N*-carbamyl-D-amino acid amidohydrolase with a novel catalytic framework common to amidohydrolases.** Takahisa Nakai, Tomokazu Hasegawa, Eiki Yamashita, Masaki Yamamoto, Tatzuo Kumasaka, Tatzuo Ueki, Hirokazu Nanba, Yasuhiro Ikenaka, Satomi Takahashi, Mamoru Sato and Tomitake Tsukihara (2000). *Structure* **8**, 729–737.
N-Carbamyl-D-amino acid amidohydrolase (DCase) catalyzes the hydrolysis of *N*-carbamyl-D-amino acids to the corresponding D-amino acids, which are useful intermediates in the preparation of β-lactam antibiotics. The crystal structure of DCase reveals a homotetramer with an unusual topology comprising a sandwich of parallel β sheets surrounded by two layers of α helices. The catalytic centre could be identified with Cys171 as the catalytic nucleophile. DCase shows only weak sequence similarity with a family of amidohydrolases, including β-alanine synthase, aliphatic amidases and nitrilases, but might share highly conserved residues in a novel framework.
20 June 2000, *Structure*